The study by Bonello *et al*[14], published in this issue of *Heart*, addresses prevention of myonecrosis using trimetazidine, an anti-ischaemic agent. In that study, 266 patients were randomly assigned to pretreatment with an oral loading dose of 60 mg trimetazidine or placebo. The group treated with trimetazidine had troponin values consistently lower than controls, and the total amount of post-PCI troponin release was reduced. This result was achieved despite a slight imbalance in baseline risk for periprocedural events against the trimetazidine group (in which patients were older, more frequently had diabetes mellitus and had a high body mass index and a high angina class). Because trimetazidine is devoid of haemodynamic effects and has overall excellent clinical tolerance, it is an attractive candidate as adjunctive treatment to PCI to decrease periprocedural myocardial injury. There are, however, limitations to this study: it is a single-centre, open study; in addition, although troponin release was reduced by trimetazidine, the frequency of troponin rise was not. Clearly, larger clinical studies powered on genuine clinical outcomes are warranted to confirm the findings of Bonello *et al* before such treatment can be adopted in clinical practice.

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PGS has been a speaker and has served on advisory boards for Servier, the manufacturer of trimetazidine.

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